of urease; all these specimens yielded a heavy growth of *C pyloridis*. One-third of tests were positive by 3 h and half by 6 h.

The advantage of this rapid test is the achievement of a diagnosis of *C pyloridis* gastritis on the same day that patients attend for endoscopy, thus avoiding a second hospital appointment. We have found this very useful for the enrolment of patients into a therapeutic trial comparing medications.

We thank our colleagues for obtaining the biopsy samples.

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CAMPYLOBACTER PYLORIDIS IN PEPTIC ULCER

SIR,—We read with interest Dr Rathbone and colleagues' letter (May 25, p 1217) in response to our April 20 report and apologise for our failure to make it clear that all peptic ulcer patients were diagnosed by endoscopy and that the sera studied were collected before treatment. The antibody assays were performed under code.

The 50 members of laboratory staff did not undergo endoscopy (for obvious reasons) nor have they been labelled as a reference group. Similar remarks apply to the children's sera, which were a general collection referred to our hospital for viral studies. The purpose of our communication was solely to report the difference found between these cohorts. The data encourage speculation as to a link between *Campylobacter pyloridis* and peptic ulcer. The raised titres in well people may be due to symptomless infection, perhaps a carrier state—this is not unknown in infectious diseases and has provided a stimulus to epidemiologically based studies.

Rathbone et al suggest that under some circumstances a specific gut IgM response might occur without either an IgA or IgG response. This is at odds with generally accepted mechanisms of gut immunity, which regard IgA as the first immunoglobulin of response in the gut.[1]

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LIFETIME PASSIVE SMOKING AND CANCER RISK

SIR,—Dr Sandler and colleagues (Feb 8, p 312) present results in their table I showing that odds ratios for overall cancer risk increase markedly in relation to the number of household members who smoke, and this increase is similar for active smokers as for non-smokers. Professor Burch (April 13, p 866) comments that this similarity leads to the paradoxical conclusion that the average effects of active smoking and passive smoking must be equal and opposite. In reply, Sandler and colleagues point out that this equality is in reality a superficial averaging of two findings—a greater odds ratio for non-smokers than smokers in relation to passive smoking as an adult, and a greater odds ratio for smokers than non-smokers in relation to passive smoke exposure in childhood. Surely, however, the latter finding is even more implausible than equality of effect in smokers and non-smokers. On any plausible model, the relative effect of passive smoking should be greater in non-smokers, who start from a smaller background level, than in smokers. Mathematically, if β is the background level of risk in the absence of passive smoke exposure and δ the increment in risk resulting from passive smoke exposure, the odds ratio $(\beta + \delta)/\beta$ will tend to be smaller the greater the value of β.

Sandler's findings are implausible in other respects—notably the large effect claimed for passive smoking for a number of cancers (breast, thyroid, leukaemia/lymphoma) that are generally believed to have little or no relationship to active smoking—and attention must inevitably centre on the adequacy of the study methods used. The choice of controls used in this study, a mixture of friends or acquaintances of patients and people randomly selected by systematic telephone sampling, is certainly unusual and seems very open to question. Sandler and colleagues admit that the study cannot be used to relate active smoking to risk of cancer, since estimates will be biased by the similarity of active smoking habits of friends. Surely, since active and passive smoking are strongly correlated, bias in studying the relationship of passive smoking to risk of cancer will also arise.

Bias may also arise because of the difference in method of approach. Thus, in a separate paper,[1] Sandler et al note that the proportion of subjects not answering questions on marital status was over three times greater in controls than in cases. If there are highly significant differences in the proportion of certain questions being answered at all, how does one know that there are not highly significant differences in the way the passive smoking questions are dealt with?

Given that active smokers receive substantial passive smoke exposure from their own cigarettes, it is a priori implausible that passive smoking should increase risk of cancers that are not associated with active smoking. Seen in this light, a much more appropriate analysis of Sandler's data would be to treat patients with smoking-related cancers as cases and patients with non-smoking-related cancers as controls. Calculations from data presented in table III of their *Lancet* paper indicate that there is no significant relation between passive smoking and cancer risk if the data are analysed in this way. This is a more plausible finding, and consistent with the results of my 1984 review[2] which concluded that there is as yet no convincing evidence that passive smoking results in any material risk of serious health hazards.

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SIR,—In their reply accompanying my letter of April 13 (p 866) Dr Sandler and colleagues misrepresent me as arguing that "smoking is protective". In fact I pointed to three possible interpretations of their findings and concluded that "active smoking has little or no net carcinogenic action". The new breakdown of their findings does not eliminate the paradoxes implicit in the aggregate data.

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BENZTROPINE INHIBITS TOXICITY OF MPTP IN MICE

SIR,—The discovery that MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) causes irreversible parkinsonism in man and other primates has provided new clues as to the cause of Parkinson's disease. The ability of MPTP to produce a relatively specific destruction of dopaminergic nigrostriatal neurons can be prevented by inhibitors of the enzyme monoamine oxidase B, including deprenyl, in primates[1,2] and mice.[3] Deprenyl (selegiline hydrochloride) has been in use in Europe for some years as an adjunct to levodopa treatment because of its ability to inhibit dopamine catabolism in the brain. Now it is suggested that early treatment with deprenyl might slow or even prevent progression of Parkinson's disease by preventing toxicity of some MPTP-like substance conceived as responsible for Parkinson's disease.

MPTP is not neurotoxic; its oxidation product MPP^+ (1-methyl-4-phenylpyridine) is.[4] MPP^+ accumulates in nigrostriatal neurons via the dopamine neuronal uptake system; MPP^+ uptake into rat striatal synaptosomes is inhibited by dopamine uptake inhibitors